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Prognostic utility of *BRAF* mutation in thyroid cancer

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Genetic alteration is the driving force for thyroid tumorigenesis and progression, based upon which novel approaches to the management of thyroid cancer can be developed. The T1799A *BRAF* mutation in papillary thyroid cancer (PTC) has a great clinical promise and is currently being translated from laboratory to the clinic use. Studies from our and other groups have consistently shown that *BRAF* mutation is the most common genetic alteration in thyroid cancer, occurring in about 45% of papillary thyroid cancers (PTC) and 25% anaplastic thyroid cancer. The *BRAF* mutation exerts its oncogenic role through aberrant activation of the MAP kinase signaling pathway. Numerous studies around the world have demonstrated the unique role of *BRAF* mutation in the development of aggressiveness of PTC, in agreement with our initial findings. For example, *BRAF* mutation is closely associated with extrathyroidal invasion, lymph node metastasis, advanced tumor stage, and, importantly, disease persistence/recurrence and even decreased patient mortality. *BRAF* mutation is also associated with loss of radioiodine avidity of PTC, making it difficult to treat this cancer using radioiodine. Numerous studies have demonstrated that *BRAF* mutation is associated with increased expression of tumor-promoting molecules or suppression of tumor-suppressing molecules, providing a molecular basis for the role of this mutation in the progression and aggressiveness of PTC. Recent studies have also demonstrated an important role of *BRAF* mutation in the silencing of iodide-handling genes in PTC, providing a molecular explanation for the association of loss of radioiodine avidity of PTC with *BRAF* mutation. Thus, *BRAF* mutation is a novel and powerful prognostic molecular marker for poorer prognosis of PTC. Use of *BRAF* mutation, which can be detected on preoperative thyroid fine needle biopsy specimens, is expected to become an effective strategy for risk stratification of PTC. This may help resolve several clinical dilemmas encountered in the management of PTC, such as how to determine the extent of surgical and medical treatments of PTC in various clinical settings. It is thus expected that *BRAF* mutation, as a novel prognostic marker in PTC, will have an important impact on thyroid cancer medicine.

Biography

Mingzhao Xing, M.D., Ph.D., is Associate Professor of Medicine, Oncology and Cellular and Molecular Medicine, Co-Director of the Thyroid Tumor Center, and Chief of the Laboratory for Cellular and Molecular Thyroid Research at the Johns Hopkins University School of Medicine. Following his initial medical training at the Second Military Medical University in Shanghai, China, he obtained a Ph. D. degree in Physiology and Biophysics at Case Western Reserve University in Cleveland. He subsequently completed an internal medicine residency at the Greater Baltimore Medical Center and a clinical fellowship in Endocrinology and Metabolism at the Johns Hopkins University School of Medicine. Upon completing the fellowship, Dr. Xing was recruited to the faculty at the Division of Endocrinology and Metabolism of the Johns Hopkins Hospital. Dr. Xing serves on a number of national and international professional committees/panels, including, for example, National Institute of Health study sections, American Thyroid Association committees, several cancer research grant review panels in European counties. He also serves as a member or editor on a number of subspecialty journals, such as Journal of Clinical Endocrinology and Metabolism, Endocrine-Related Cancer, and Thyroid. Dr. Xing practices clinical endocrinology as a subspecialty consultant and teaching attending at the Johns Hopkins Hospital while also conducting laboratory research as a physician scientist. His main clinical and research interest is in thyroid diseases, particularly thyroid tumors. Supported by the American Cancer Society and NIH R0-1 grants, his laboratory has been studying molecular, genetic and epigenetic mechanisms of thyroid cancer and their clinical translations. His team has published actively in these areas, particularly in relation to the MAP kinase and PI3K/Akt pathways. He is co-holder of a patent on the initial discovery and clinical characterization of the BRAF mutation in thyroid cancer. He has published more than 80 scientific articles. Among his professional recognitions/awards are the US FAMRI Clinical Innovator Award, Maryland Innovator Award, American Cancer Society RSG Award, and “America’s Top Physician” recognition.